Use of pyridoxine supplements in pregnancies associated with glucose intolerance

G. Vijaykumar, C.V. Krishnaswamy, J.R. Subramanyam, M. Chellamariappan, P.V. Ashabai, K. Balamurugan, P. Krishnaswamy, M. Dhamija, S. Fernando, Vijayalakshmi, V. Prasad, B. Subramanyam, S. Suresh

ABSTRACT

Out of 1262 deliveries conducted between March, 1988 and September, 1990, 44 patients (3.84%) having diabetes mellitus or impaired glucose tolerance were enrolled for the study. All patients in this study group were treated with 80 mg of pyridoxine per day orally. Nature of glucose intolerance, degree of metabolic control, obstetric history, timing and mode of delivery and foetal outcome were documented. No foetus in this group suffered from congenital anomalies as compared to 2.3% of anomalies seen in non-diabetic deliveries.

This study raises pyridoxine supplementation as an interesting therapeutic possibility in pregnancies associated with glucose intolerance.

INTRODUCTION

Coelingh Bennink et al (1) described an increased excretion of xanthurenic acid in the urine of gestational diabetics. Pyridoxine supplementation caused a decrease in the urinary excretion of xanthurenic acid and an improvement in the oral glucose tolerance test. They postulated that pyridoxine restored the tryptophan metabolism to normal. We conducted this study to confirm the findings of these investigators.

MATERIAL AND METHODS

The study period extended between March 1988 and September 1990. During this period a total of 1262 deliveries took place, out of which 44 (3.48%) patients suffered from diabetes mellitus or impaired glucose tolerance. The expectant mothers who were in the high risk group, and those pregnant women who had the random blood glucose of more than 120 mg/dl were subjected to oral glucose tolerance test with 75 gms glucose. Depending on these results two groups were categorised: 1. Overt Diabetes 2. Abnormal Glucose Tolerance (AGT). Our criteria in categorising these cases are as follows:

1. Overt Diabetes: When GTT showed peak and 2 hour value ≥ 180 mg%.

2. Abnormal Glucose Tolerance: peak ≥ 160 mg%, and 2 hour value ≥ 120 mg% and < 180 mg%.

With this, total number of cases taken for the study, were 44. Amongst these, number of cases with overt diabetes were 20, 11 had diabetes prior to conception (4 NIDDM and 7 IDDM). 21 had abnormal glucose tolerance and 3 had renal glycosuria. The detailed obstetric history of these cases had been taken. Out of the total 44 cases, 12 (27.2%) were primi gravidae and in the remaining 32 who were multi-gravidae, 23 (72%) had bad obstetric history (9 had history of intra uterine death, 1 had premature delivery, 15 had abortions). Only one patient had history of delivering a congenitally anomalous baby (anencephaly). 70% of these cases had a positive family history for diabetes mellitus.

Treatment requirements:

All these cases were put on diet therapy and tablet pyridoxine 80 mg per day. 11 cases had diabetes prior to conception and all of them required insulin. In the remaining 33 cases only 4 (12%) required insulin.

Metabolic control:

The metabolic control was with frequent assessment of blood sugar, GHb every 2 to 3 months and serum frutosamine every 3 to 4 weeks. 24-hour blood sugar profiles were done when required either by hospitalization or by home blood glucose monitoring.

The parameters studied and the results were:
PPBS (Mean ± SD): 145.85 ± 45.05 mg%
GHb- Assayed by column chromatography (Mean ± SD): 9.01 ± 1.23 %

From: Department of Diabetes, Voluntary Health Services Medical Centre, Madras-600 113. India.

Fructosamine- Assayed by colorimetric method (Mean ± SD): 2.51 ± 0.75 mmol/L

Normal range: 1.6-2.6 mmol/L
Satisfactory control: 2.6-3.2 mmol/L
Mediocre control: 3.2-3.7 mmol/L
Poor control: 3.7-4.5 mmol/L

Mode of delivery:

79% of the cases were delivered by LSCS and 21% vaginally. The mean gestational age at delivery was 38.2 weeks (SD= 1.63).

RESULTS

40 cases (91%) had successful outcome. Average birth weight of the baby was 3.28 kgs. None of the cases had congenital anomalies (including babies, which were lost). Congenital anomalies seen in non-diabetic pregnancies in this study was 2.3%. In a study in 1986, by one of us (C.V. Krishnaswamy), out of a total of 48 diabetic deliveries there were 6 neonatal and perinatal mortalities; out of this 3 cases had congenital malformations, and the rest due to infections.

It is therefore evident from our experience that the incidence of congenital malformations in pyridoxine-treated patients is not significantly greater than in non-diabetic deliveries. It is also found that intrauterine deaths were more frequent in our conditions and efforts to prevent this are to be made and also to avoid perinatal mortality by means of better neonatal care. The causes for the 4 unsuccessful deliveries in this study were found to be due to obstetric and reasons other than diabetes.

CONCLUSIONS

Treatment of pregnant diabetics with pyridoxine reduces the incidence of congenital malformations. In the group under study, no malformation was observed.

In a similar group of 48 patients reported by one of us earlier, congenital malformations occured in 3 patients.

REFERENCES

1. Bennink HJTC and Schreurs WHP. Improvement of OGTT in GDM after pyridoxine. British Medical Journal 1975;3:13.